

Jim Lucas -

US EPA RECORDS CENTER REGION 5



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Here is a copy of the cooperative agreement application from the University of Minnesota, School of Public Health. Also attached is a report and some data which I have put together which may be of assistance in the evaluation of need for such a project.

Essentially I have been pursuing two areas of research concerning PAH contamination of the municipal drinking water supply in St. Louis Park, Minnesota. The first is exposure assessment. Studies have been designed to determine the high exposure areas within SLP. This increases the sensitivity of future human health assessment studies. I have been in active cooperation with the Minnesota Department of Health concerning these projects. The second area of interest is to determine if the SLP population has unreasonable cancer incidence. That is the gist of the cooperative agreement with Minnesota's School of Public Health.

The value of pursuing these projects at this time stem from both a 'Public Health' and 'research' concern. It is presently not known what effects on human health these exposures have. It is noteworthy that the SLP population is still exposed to significant levels of PAH in their drinking water. This is especially true during the summer months. If we wait until there is no current exposure, we may lose the capability to evaluate the relationship between exposure, dose, metabolic effect, and human health effect. There also exist good data concerning the mutagenicity of PAH in bacterial assay systems. carcinogenicity in animal (multiple species, dose rates, and routes of exposure) bioassay, and suggestive evidence for carcinogenicity from occupational exposure. The addition of data from environmental exposure at these levels could prove to be very interesting.

These investigations have relevance to the Health Effects Research Laboratory (Drinking Water), Toxics and Hazardous Waste, Standards and Criteria, and Exposure Assessment Methodology within the agency. Other interested agencies could include National Institute Occupational Safety and Health, Department of Energy (Synfuel and Coal Utilization), National Cancer Institute; and International Agency for Research on Cancer.

Jack Sahi

Polynuclear Aromatic Hydrocarbons in Drinking Waters
Human Health Effects of Chronic Low Level Exposure

Report and Study Proposals

Jack D. Sahl

Health Effects Research Laboratory
Chronic Disease and Biostatistics Branch
United States Environmental Protection Agency

Contacts :

Minnesota Department of Health
717 Delaware Street S.E.
Minneapolis, Minnesota 55440

Mr. David Giese
Assistant to the Director
Division of Environmental Health
(612) 296-5362
(FTS) 776-5362

Mr. Gary Englund
Chief, Water Supply & Engineering
Division of Environmental Health
(612) 296-5330
(FTS) 776-5330

Jim Nye - Environmental Scientist
Tony Manoukian - Environmental Scientist
(612) 296-5554
(FTS) 776-5554

Mr. Michael Convery
Hydrologist
(612) 296-5297
(FTS) 776-5297

Dr. Dave Gray - Chief, Health Risk Assessment
Dr. Ingrid Riche - Health Risk Assessment
Dr. Andrew Dean - Director, Division of Epidemiology
Dr. Alan Binner - Epidemiologist

University of Minnesota
School of Public Health
Minneapolis, Minnesota 55455

Professor Rexford Singer
Acting Director, Department of Environmental Health
(612) 373-8080
(FTS) 786-8080

Dr. Jack Mandel
Assistant Director, Department of Epidemiology
(612) 373-8030
(612) 373-8028
(FTS) 786-8030

United States Environmental Protection Agency

Mike Kosakowski
Office of Hazardous Waste Enforcement
U.S. EPA
Washington, D.C. 20460
MR: EN 335
(FTS) 472-3500

Jim Pankanin
Enforcement Division
U.S. EPA
230 S. Dearborn Street
Chicago, Illinois 60604
(FTS) 886-6769

Polynuclear Aromatic Hydrocarbons in Drinking Waters

Human Health Effects of Chronic Low Level Exposure

Several polynuclear aromatic hydrocarbons (PAH) have been shown to be tumorigenic in animal bioassay and have been associated with occupational carcinogenesis. These compounds are ubiquitous in air, soil, food, and water. World Health Organization (WHO) recommends that the sum concentration of six specified PAH in drinking waters be limited to 200 ng/l. Basu and Saxena samples ten U.S. municipal water supplies for PAH and found a range of 0.9 ng/l to 15ng/l. U.S. EPA criteria document recommends that BaP water concentration be maintained below 28 ng/l.

St. Louis Park (SLP) is a 48,000+ (1970) municipality in Minnesota. Extensive soil and groundwater contamination with coal-tar by-products, creosote, and their waste products has occurred due to operations by Reilly Tar and Chemical from 1917 to 1972. In November 1978, four drinking water wells were closed due to heavy PAH contamination (120-1,500 ng/l), two of which had 300 ng/l fluoranthrene. Six wells currently in use average 50 ng/l total PAH. Duration of municipal drinking water contamination is not known. It is not known what significance to human health these levels pose.

Total PAH levels in SLP wellwater were the highest reported for a municipal water supply. Using national ambient levels as background exposure (total PAH .442-1.878 ug/day), the SLP population may have been exposed to PAH from 31% to 678% of their average total exposure from their drinking water supply.

Human health effects of exposure to ambient PAH is of interest. Combustion is the predominant end process by which fossil fuels are converted to energy and this is the primary source of environmental PAH. With the current and projected energy base of the U.S. we can expect ambient PAH levels to increase.

Drinking water supplies may become contaminated with PAH through improper disposal of hazardous wastes, urban run-off, municipal sewage, and washout. With increasing real and potential contamination with these compounds it becomes important to assess human health risks associated with these exposures. SLP municipal drinking water contamination provides an outstanding opportunity to adequately study the presence or absence of increased human health risk due to chronic low level PAH exposure. Additional epidemiologic studies in SLP would provide data to assess these risks. These data would enhance public health knowledge of population risk due to ambient PAH exposure. Thus additional investigations are warranted.

Polynuclear Aromatic Hydrocarbon Study Proposals and Sequence

- 1). Retrieve and evaluate information and data currently available relevant to SLP contamination history. Additionally, data specific to SLP hydrology, municipal drinking water supply system characteristics, and demography needs to be abstracted.
- 2). Literature review of the sources, occurrence, and potential human health effects due to environmental exposure to PAH.
- 3). Provide assessment of the human health implications of the drinking water contamination, direction(s), and priority of further studies.

Exposure Assessment

- 4). Abstract water well pumping records and water supply distribution system data.
- 5). Water well service area assessment study. Utilize surrogate water marker parameter to indicate average well service area using historical pumping records as normal operating conditions.
- 6). PAH analysis of SLP distribution system water to provide data on current population exposure and water treatment effects on PAH levels.

Metabolic Effects

- 7). Select biological samples from SLP residents within exposure area and compare with non-drinking water PAH exposed individuals.
Option 1- Bioaccumulation in body tissue;
Option 2- PAH enzyme induction (AHH) as indicator of exposure.

Human Health Effects

- 8). Review Third National Cancer Survey (TNCS) data to determine if a greater than expected incidence occurs in SLP.
- 9). Review TNCS data by census tract to determine if clustering occurs within SLP due to varying exposure potential.
- 10). Surveillance of cancer incidence (1979-1981) in SLP to compare with TNCS data.
- 11). Case/Control population based cancer incidence study keying on relevant tissue endpoints.
- 12). Infant birthweight record study to investigate if chronic maternal exposure to PAH in drinking water effects average population birthweight.

Polynuclear Aromatic Hydrocarbons in Drinking Waters:
Human Health Effects of Chronic Low Level Exposure

St. Louis Park (SLP) is a 48,000+ (1970) municipality in the Minneapolis-St. Paul (Mpls-St Paul) Standard Metropolitan Statistical Area (SMSA), Minnesota. Extensive soil and ground water contamination with coal-tar by-products, creosote, and their waste products has occurred on an 80 acre site formerly occupied by Reilly Tar and Chemical Corporations (35, 36). Reilly operated between 1917 and 1972. Soil contamination has occurred due to improper disposal of wastes. Groundwater contamination has occurred due to infiltration of spills and seepage; aquifer recharge from ponds receiving plant run-off and contaminated process water; and multi-aquifer wells becoming contaminated (30, 42).

Polynuclear aromatic hydrocarbons (PAH) comprise a significant fraction of coal-tar and creosote solutions (Table 1). SLP municipal drinking water supply has been shown to be contaminated with phenolic and PAH compounds (27, 36).

Mpls-St Paul SMSA has a largely white (+98%), high social-economic status, Northern European population. SLP is similar in these respects. 1970 demographic census also indicates a stable population with 20% Jewish, high educational achievement, and smaller average family size for SLP when compared to Mpls-St Paul SMSA. (Census Data)

Human health effects of exposure to ambient PAH is of interest for a number of reasons. Combustion is the predominant end process by which fossil fuels are converted to energy and this is the primary source of environmental PAH (19, 59, 75). Guerin (33) has stated that "PAH related carcinogenesis is among the most important of possible occupational and environmental health impacts of much of the current and anticipated energy base." Examples include the observation that a single commercial coal liquidification plant will produce 120,000 kg PAH and 7 kg benzo(a)pyrene (BaP) daily as a component of the product (33). The processing of 10^6 tons of coal per day would produce approximately 100 metric tons BaP annually (33). Thus we can expect ambient levels to increase with increased use of coal and synthetic fuels. PAH compounds may also be present in drinking water supplies due to the addition of protective paints and coatings to distribution and storage systems.

Table 1.

Polynuclear Hydrocarbons in Creosote and Medicinal Coal-Tar (51)

	Conc in Creosote (g/kg)		Conc in Coal Tar (g/kg)	
	(1)	(2)	(1)	(2)
Anthracene	12.1	12.0	2.88	4.35
Benz(a)anthracene	2.77	2.94	6.24	6.98
Benzo(b)chrysene	0.03	0.06	0.93	0.80
Benzo(a)pyrene	0.14	0.22	2.08	1.76
Chrysene	1.34	0.94	2.13	2.86
Fluoranthene	24.8	22.2	17.7	17.8
Pyrene	9.1	6.8	7.95	10.5
Benzo(a,h,i)perylene	--	--	1.23	1.89
Dibenz(a,h)anthracene	--	--	0.30	0.23

Table 2. Aqueous Solubility of Polynuclear Aromatic Hydrocarbons (83)

Compound		Solubility (25° C) mg/liter	
Fluorene	C ₁₃ H ₁₀	C ₁₃ H ₁₀	1.98 ± 0.04
Phenanthrene		C ₁₄ H ₁₀	1.29 ± 0.07
Anthracene		C ₁₄ H ₁₀	0.073 ± 0.005
9, 10-Dimethyl-anthracene		C ₁₆ H ₁₄	0.056 ± 0.0005
Pyrene		C ₁₆ H ₁₄	0.135 ± 0.005
Fluoranthene		C ₁₆ H ₁₀	0.26 ± 0.002
Chrysene		C ₁₈ H ₁₂	0.0020 ± 0.00017
Naphthacene		C ₁₈ H ₁₂	0.00057 ± 0.00003
7,12-Dimethyl-1,2-benzanthracene		C ₂₀ H ₁₆	0.061 ± 0.0006
3,4-Benzopyrene		C ₂₀ H ₁₂	0.0038 ± 0.00031
Benzo (g,h,i)perylene		C ₂₂ H ₁₂	0.00026 ± 0.00001

Toxicology and Human Health

The human toxicologic effects of PAH have not been adequately qualified or quantified. Most PAH toxicologic investigations have studied BaP with animal bioassay and cell culture systems. Many questions remain unanswered at this time. These include: 1) What are the health effects to human populations when chronically exposed to low levels of potentially carcinogenic compounds in a drinking water supply?; 2) Do these compounds act as promoters or co-carcinogens in human populations at the levels encountered?; 3) Does there exist a level to which human populations can be exposed to potential carcinogens without developing cancer?; and 4) Does exposure to low levels of PAH induce the microsomal mixed-function oxidase (MFO) system, increasing the metabolism of these compounds, thus increasing their toxic effect?

Pharmacokinetics

Absorption of PAH occurs across epithelial membranes due to high lipid solubility. Evidence exists that PAH are easily absorbed through the lung after inhalation (65) and that intestinal passive diffusion also occurs (62). PAH are stored primarily in body fat and fatty tissue (8, 69, 70). Benzo(a)pyrene (BaP) is rapidly eliminated from blood and liver to body fat (18). Pretreatment with BaP (inducing microsomal activity) increases the rate of metabolism in all tissues (70). Pharmacological implications of inducing microsomal enzyme activity is discussed by Conney (20).

PAH require enzyme bioactivation prior to initiating toxic effects (23). Metabolism occurs thru enzyme-mediated oxidative mechanisms forming electrophiles (49). Primary metabolism occurs with the cytochrome P-450-dependent microsomal mixed-function oxidase (MFO) system, also designated as aryl hydrocarbon hydroxylase (AHH) (20, 31, 74). The MFO system metabolises endogenous and xenobiotic substances and is present in most mammalian tissues (17, 76). The quality and quantity of PAH metabolizers will vary depending upon species, nutritional status, sex, age, and prior exposure to enzyme-inducing chemicals (23, 53).

The rate of excretion and metabolism are influenced by structure of the parent compound and route of exposure (1, 2) plus previous exposure to inducers of microsomal enzymes (69, 70). Adequate pharmacokinetic studies of PAH have not yet been performed. It is not yet known the extent of mammalian bioaccumulation after chronic exposure to PAH. There are indications of significant storage in body fat (37, 38) and this is supported by the high lipid solubility of PAH. However, it has been suggested that PAH will not bioaccumulate due to their capacity to induce their own metabolism, thereby increasing their water solubility and eventual excretion (77). Bioaccumulation and metabolism may be dependent on the conditions with which the animal is exposed. PAH compounds administered in different vehicles have shown different absorption, metabolism, and excretion rates (48). This would also be associated with the quantity and quality of the cytochrome P-450-dependent microsomal MFO system in cell and tissue types. It has been reported that farm animals fed BaP in their diets do accumulate BaP in their tissues (84).

Due to large intra-and interspecies variability in tissue levels of the MFO enzyme system, and the relative amount of body fat in an individual, the levels of storage or bioaccumulation could be expected to be an individual characteristic.

Human Health Effects

Only a small percentage of PAH compounds are thought to be carcinogenic. Thus, total PAH measurements need to be qualified and cannot necessarily be equated with carcinogenic risk. Carcinogenic PAH are thought to include: benzo(a)pyrene; benzo(a)anthracene; benzo(j)fluoranthrene; dibenz(a,h)anthracene; benzo(b)fluoranthrene; chrysene; dibenzo(a, h)pyrene; dibenzo(a,i)-pyrene; dibenzo(a,l)pyrene; indo(1, 2, 3-cd)pyrene; 7, 12-dimethyl benzanthracene; and methylcholanthrene (19, 28). Noncarcinogenic PAH (pyrene, benzo(e)pyrene and benzo(a,h,i)perylene) demonstrated a cocarcinogenic effect when administered in low doses with BaP (78, 79). Anthracene (71) and naphthacene (16) are also potential cocarcinogens or promoters. An immunosuppressive effect has also been observed to correlate with PAH carcinogenic potency (5). Excess mutagenicity with the salmonella/microsome bioassay for a variety of PAH has been found (54).

Carcinogenicity of various PAH (predominately BaP) have been ascertained by intragastric (41); intravenous; intramuscular; skin painting; aerosol, oral (64); and dietary exposure in a number of mammalian species (65, 19, 28). Leukemias, adenomas, sarcomas, and papillomas have developed in the test species (19, 24). Tissues include lung, skin, breast, liver, and stomach in various species with a wide range of dose (19, 24). The carcinogenic potency of PAH are highly variable. This plus dose have a great effect on the latency of tumorigenesis (19, 24).

BaP has been shown to be an effective carcinogen at chronic low doses and at acute doses (40). Payne and Hueper (57) concluded that for mice, a given amount of carcinogen is more effective when administered over a long period rather than as a single dose. Suggesting that a low-level, recurring exposure to a carcinogen may be more hazardous than a single exposure to the same amount (67).

Antioxidants are effective inhibitors of PAH-induced tumor development, Selenium (72), vitamins E and C, and food preservatives BHT and BHA have inhibited lung, breast, and gastric tumor formation (73, 80). Decreases in dietary protein also decreased the activation of carcinogenic PAH (21) as did presence of vitamin A (56).

Limited data are available concerning teratogenic effects of PAH in humans or other animals. Rigdon and co-workers (63, 64) investigated teratogenic effects of BaP feeding in rats and mice. One malformed fetus out of seven litters maternally exposed was found. An excess of reabsorptions and dead fetuses was also apparent. Mothers were fed 1 mg/g BaP. Bulay (13) and Bulay and Watterberg (14) reported inconclusive results for teratogenic effects in mice.

Cigarette smoking has been associated with low infant birthweight (61). The PAH content of cigarette smoke has been implicated in this effect. PAH compounds induce aryl hydrocarbon hydroxylase activity in mammalian placenta (9, 46, 55, 81). This early induction may permanently alter PAH metabolic capability in individuals so exposed (29). The health consequences of these effects are not known.

Environmental exposure of PAH is most likely to occur as a complex and undetermined mixture. Ambient exposure will occur due to ingestion and inhalation. How this complex mixture of routes of exposure and compounds affects human health is not known at this time.

Epidemiologic Human Investigations

Percivall Pott found an association between scrotal cancer and chimney sweeps (58). Leitch (50) observed an association between skin cancer and chimney sweeps and later the PAH BaP was shown to be a constituent of soot (32). Skin cancer is presently associated with the coal-tar, oil, tanning, and chemical occupations (28, 39).

Respiratory cancer has been found in occupationally exposed coke-oven workers, gas workers, roofers and water proofers (28, 60). Hammond (34) found that an air exposure of 6,000 ng/m³ of BaP was associated with cancers of the lung, buccal cavity, pharynx, larynx, esophagus, bladder, and stomach. Studies by Doll *et al.* (25) with coal carbonization workers; Kawai *et al.* (47) with generator gas plant workers; and Lloyd (52) with steelworkers confirmed an increased risk of cancer, largely thought to be a result of increased PAH exposure.

No epidemiological studies have been performed of environmentally exposed human populations to PAH compounds.

Ambient Environmental PAH Exposure

Humans can be environmentally exposed to PAH thru air, food, and water.

World Health Organization (WHO) International Agency for Research on Cancer (IARC) has not been able to estimate the hazard to man from PAH encountered in the ambient environment. Although these levels are less than those encountered in occupational settings, synergistic effects of other substances, and chronic low level exposure may increase risk. Without sufficient data in this area no prediction of human health risk can be made from knowing ambient PAH concentrations.

Air

PAH concentrations in ambient air are dependent upon a number of factors. Levels appear to be the same indoors as outdoors in suburban areas (15). Levels are lowest during summer months and highest during winter months. This is somewhat a source effect, with an increase in space heating during winter months. Meteorological conditions can affect concentrations due to transport and/or fallout. Ambient air levels appear to be decreasing in U.S. urban areas (68).

Dietary

PAH concentrations in uncooked foods mainly depend on the source of food. Foodstuffs from polluted environments can be expected to contain higher concentrations of PAH. In the case of cooked foods, the method of cooking is largely responsible for the PAH content of the food. Longer cooking time, closer position to heat source, and allowing fat to drip onto heat source tend to increase PAH in cooked foods. Smoking of foods and char broiling has been associated with high levels of dietary PAH. Dietary exposure levels are subject to regional and cultural variations (19, 24, 28).

Drinking Water

PAH were analyzed for in some U.S. drinking water supplies (3, 4, 6, 7). Generally levels were lower than 15 ng/l. European groundwater levels normally will be lower than 50 ng/l with 100 ng/l found in some drinking waters (11). Analysis were performed on the six PAH recommended by WHO for drinking water monitoring (82). Surface waters can be expected to have higher levels of PAH than groundwaters, with industrial discharge and water- or air-shed runoff being significant contributors. Treatment processes can lower PAH levels in drinking water supplies. Efficiency of a treatment process or sequence being dependent on initial levels and plant operation procedures (4). It has been recommended that PAH levels in drinking waters be limited at 200 ng/l when measuring for six specified PAH compounds (11, 82).

Difficulties exist in estimating human exposure to total PAH thru environmental media. These include a lack of analytical data for the environment of interest and numerous assumptions concerning routes of exposure, absorption efficiency, population lifestyle homogeneity, and sex- and age-specific influences. Estimates of exposure are provided for air, dietary, and drinking water in table 3.

Table 3. (77)

Estimate of Human Exposure to Ambient PAH

<u>Source</u>	<u>Estimated Exposure (ug/day)</u>		Total PAH
	BaP	Carcinogenic PAH	
Water	0.0011	0.0042	0.027
Dietary	0.160-1.6		0.251-1.6
Air	0.0095-0.0435	0.03-0.046	0.164-0.251
Total	0.1695-1.6446	0.0342-0.0502	0.442-1.878

Water Treatment Effects on PAH Concentrations

It is suggested that one-third aqueous PAH is bound to suspended particles, one-third to finely dispersed particles, and one-third dissolved (4). Effectiveness of drinking water treatment will vary depending upon which fraction is being studied. Large particle bound PAH would require sedimentation, flocculation, or filtration. Dissolved PAH would be best removed by chemical oxidation thru ozonation and chlorine dioxide. Absorption by activated carbon is also an effective treatment process.

Most experiments have utilized BaP to monitor for PAH removal efficiencies. Generally, with greater sedimentation of solids we could expect higher water quality. However, the PAH bound particles are thought to be too small to settle in most clarification processes (12). Ozonation, UV and gamma irradiation, chlorination and ClO₂ treatment have been used to treat PAH in drinking waters (3, 4, 43, 44, 45, 85). Results indicate that ozonation and ClO₂ are most effective with other processes being ineffective.

By combining mechanical and chemical processes up to 99% removal efficiencies have been achieved (4, 7, 10). It has not yet been possible to reasonably treat water to reduce PAH to below 10 ng/l (ppt) (3).

Distribution of finished water may affect PAH concentration. Borneff (12) reports 10x increase in PAH concentration from contacting a pipe coating. U.S. distribution system mains generally use either cast/ductile iron or asbestos cement pipe with an unknown effect on PAH concentrations (3, 4). Those systems which have utilized protective paints or coatings are difficult to identify due to variability in products and lack of adequate record keeping.

St. Louis Park Municipal Drinking Water Supply

100% groundwater supplied with multiple wells entering throughout the distribution system. Wells are pumped on demand and directly feed an open, interconnected distribution system. Five storage reservoirs are utilized. A 1.5 million gallon surface reservoir is served by wells 3,10,11, and 15. Well 5 serves a 1,000,000 gallon elevated tank; wells 4,6, and 12 serve a 500,000 gallon elevated tank. An additional 100,000 gallon tank is located between wellfields 4,6,12 and 3,10,11, and 15 (Table 4).

SLP wells utilize Prairie du Chien aquifers (St. Peter, Shakopee, and Oneota formations). This is a dolostone and poorly cemented sandstone group and is the principle aquifer of Mpls-St Paul SMSA.

Treatment includes fluoridation and chlorination at the well head, plus some additional sand filtration to control iron. Water quality is considered good, with iron levels at 0.5-3.5 mg/l. Water supply is plentiful (see Map 1).

St. Louis Park Municipal Water Supply Contamination History

From May 1978 to August 1978 samples from municipal drinking water wells in SLP and surrounding communities were analysed for seven PAH compounds. SLP wells 10 and 15 had heavy contamination with total PAH at 1,500 ng/l for well 15 and 1,000 ng/l for well 10. Moderate contamination was found in wells 7 (120 ng/l) and 9 (230 ng/l) for total PAH (Table 5). Anthracene, pyrene, and fluoranthrene were the most significant contributors to these levels. In December 1979 well 4 was closed with total PAH around 300 ng/l (biphenyl, phenanthrene, fluorene). These wells are in the Shakopee-Jordan formation and range 446-503 feet deep. Well 3 was closed in 1932 due to phenolic taste and odor complaints.

The normal flow of groundwater in this area is Eastward. Wells 10,15,7, and 9 are North of the site of contamination. Well 4 is Southeast of the site. It has been proposed that the contaminants have been drawn to these wells through fractures in the formation due to high pumping rates. Thus well four became contaminated only after 7,9,10, and 15 were closed. This is significant in that only a year elapsed for well 4 to show contamination after 7,9,10, and 15 ceased pumping.

Of wells still in service as water supply wells, 3,5,6,11,12, and 14 have low level contamination averaging around 50 ng/l. Wells 8,13, and 16 show no contamination. In 1980 wells 8,13, and 16 were the major producers at around 12 million gallons per day each. Wells 3,5,6,11, and 12 pumped at around 8 million gallons per day each.

Of the wells still in service, 5,6,8,14, and 16 range from 465-507 feet; 11,12, and 13 range 1045-1095 feet; and well 3 is at 286 feet. Wells at 450-550 feet will possibly become contaminated in the future due to high soil contamination.

Table 4.

ST. LOUIS PARK MUNICIPAL WATER WELL PUMPAGE DATA 1970 - 1978

	<u>3-10-11-15</u>	<u>4</u>	<u>5</u>	<u>6</u>	<u>7-9</u>	<u>8</u>	<u>12</u>	<u>13-14</u>	<u>16</u>
January	2.91x10*8	6.46x10*6	1.05x10*8	2.91x10*8		1.43x10*8	2.87x10*7	1.26x10*8	1.18x10*8
February	2.99x10*8	1.46x10*7	4.28x10*6	1.69x10*8		1.42x10*8	1.97x10*7	9.79x10*7	9.64x10*7
March	3.34x10*8	2.54x10*7	1.37x10*7	1.88x10*8	4.12x10*4	1.65x10*8	1.40x10*8	1.40x10*8	1.10x10*8
April	2.74x10*8	3.48x10*7	3.24x10*7	9.72x10*7	1.95x10*6	1.98x10*8	2.88x10*8	1.23x10*8	8.71x10*7
May	4.08x10*8	1.32x10*8	7.34x10*7	2.07x10*8	4.12x10*7	2.58x10*8	1.27x10*8	2.68x10*8	1.16x10*8
June	9.17x10*8	1.63x10*8	1.90x10*8	2.35x10*8	2.29x10*8	5.71x10*8	2.58x10*8	3.47x10*8	1.76x10*8
July	5.53x10*8	1.11x10*8	1.40x10*8	2.80x10*8	1.45x10*8	3.27x10*8	1.65x10*8	2.37x10*8	7.63x10*7
August	4.82x10*8	1.06x10*8	9.34x10*7	2.24x10*8	5.81x10*7	3.39x10*8	2.04x10*8	5.06x10*7	1.22x10*8
September	3.79x10*8	8.12x10*7	8.67x10*7	1.21x10*8	4.16x10*7	2.71x10*8	7.18x10*7	2.03x10*8	1.13x10*8
October	2.01x10*8	4.33x10*7	8.16x10*7	1.70x10*8	4.06x10*7	2.59x10*8	5.71x10*7	2.42x10*8	1.17x10*8
November	2.39x10*8	2.67x10*7	4.71x10*7	1.71x10*8	2.73x10*7	2.05x10*8	7.50x10*7	1.98x10*8	1.21x10*8
December	2.73x10*8	5.83x10*7	3.04x10*8	1.50x10*8		1.69x10*8	9.75x10*7	2.19x10*8	7.23x10*7
Total	4.65x10*9	8.03x10*8	8.98x10*8	2.30x10*9	5.85x10*8	3.04x10*9	1.53x10*9	2.24x10*9	1.32x10*9
% Usage	(26.8%)	(4.6%)	(5.1%)	(13.3%)	(3.3%)	(17.5%)	(8.8%)	(12.8%)	(7.6%)

: Data is gallons per month.

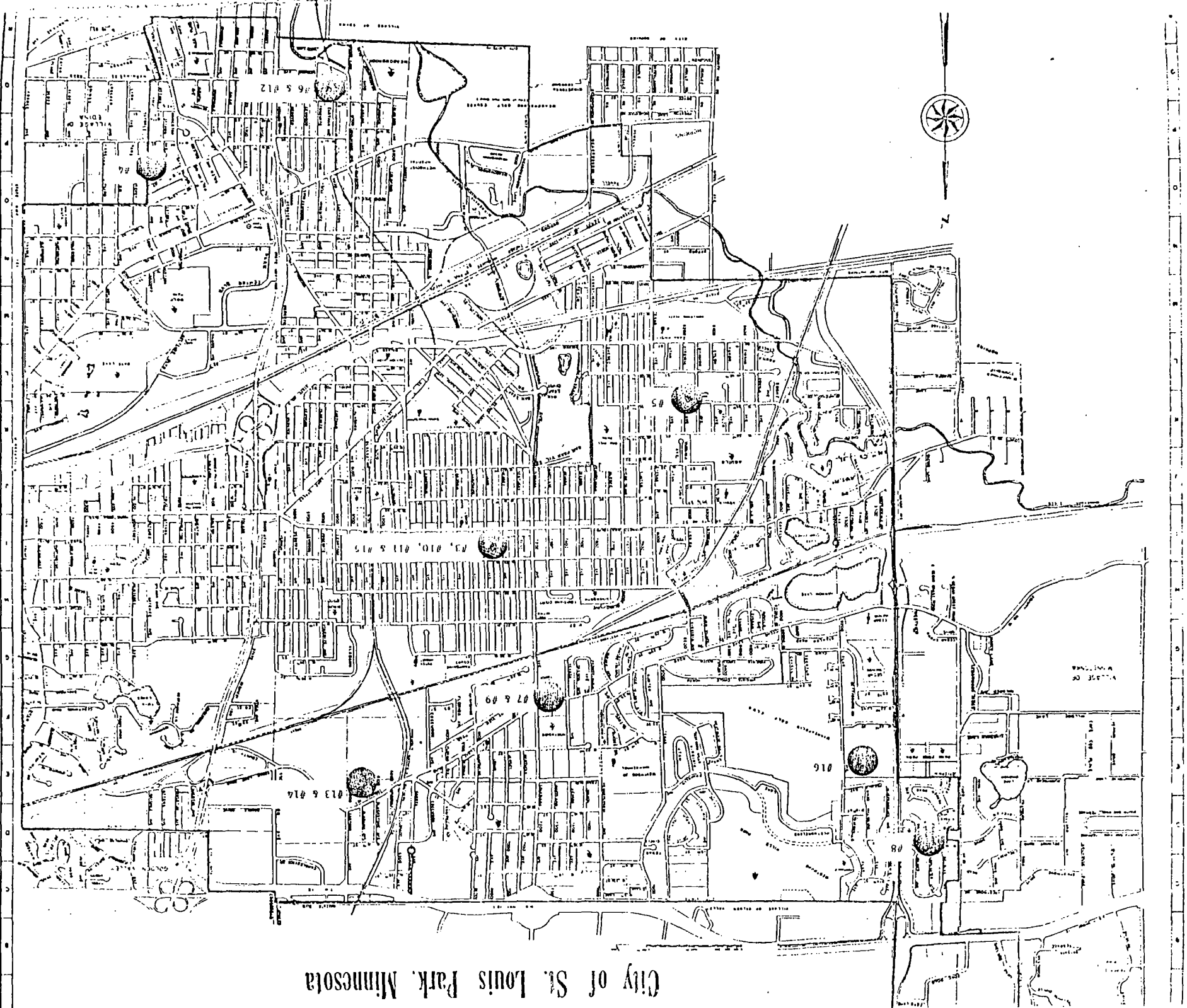
: The Expression '10*' = 10 to the n power.

Table 5 (36): PAH in St. Louis Park Drinking Water, 1978.
(ng/l, ppt)

<u>Well</u>	<u>Depth (feet)</u>	<u>A</u>	<u>P</u>	<u>FI</u>	<u>BaP</u>	<u>BghiPE</u>	<u>OPP</u>	<u>N</u>
SLP #3	236	< 1.9	< 47	< 0.9	< 1.1	< 4.4	< 1.1	< 10
SLP #4	500	< 1.9	< 47	4.5	< 1.1	< 4.4	< 1.1	< 10
SLP #5	465	< 1.9	< 47	< 7.4	< 1.0	< 4.1	< 1.2	< 10
SLP #6	480	< 1.9	< 47	< 1.0	< 1.0	< 4.8	< 1.5	< 10
SLP #7	446	11.4	104	7.4	< 1.1	< 4.4	< 1.1	< 10
SLP #8	507	< 1.9	< 47	< 0.8	< 1.1	< 4.4	< 1.1	< 10
SLP #9	473	12.2	199	21.1	< 1.1	< 4.4	< 1.1	< 10
SLP #10	500	100	800	450	< 1.1	< 9.8	< 2.1	-
SLP #10	500	54	486	152	1.3	4.4	< 1.2	80
SLP #11	1000	< 1.9	< 47	< 0.9	< 1.1	< 4.5	< 1.1	< 10
SLP #12	1095	< 1.9	< 47	< 0.9	< 1.1	< 4.5	< 1.1	< 10
SLP #13	1040	< 1.9	< 47	1.0	< 1.1	< 4.5	< 1.2	< 10
SLP #13	1040	< 1.9	< 47	< 0.9	< 1.2	< 4.9	< 1.1	< 10
SLP #14	485	6.3	< 47	4.2	1.8	5.5	2.2	< 10
SLP #14	485	6.3	< 47	2.4	< 1.2	5.4	< 1.1	< 10
SLP #15	503	190	750	390	< 1.2	< 10.7	< 2.4	-
SLP #15	503	241	1221	292	1.5	6.8	2.0	160
SLP #16	500	< 1.9	< 47	< 0.8	< 1.1	< 4.4	< 1.1	< 10

Key: < = less than detection limit. A = anthracene, P = pyrene, FI = fluoranthene, BaP = benzo(a)pyrene, BghiPE = benzo(a,h,i)perylene, OPP = O-phenylenepyrene, N = naphthacene.

City of St. Louis Park, Minnesota



MUNICIPAL WELL DATA - ST. LOUIS PARK, MINNESOTA

Number & Address	<u>Date</u>	<u>Well Depth</u>	<u>Casing</u>	<u>Openhole</u>	<u>Grouted</u>	<u>Treatment</u>
NON-RESPONSIVE	1939	286'	24" to 103'	103' - 286'	?	Chlorination Aeration Sand Filtration
	1946	503'	24" to 90' 18" to 304' 12" to 415'	415' - 503'	415'	Chlorination
	1947	465'	24" to 115' 20" to 305'	305' - 465'	305'	Chlorination
	1948	480'	24" to 107' 20" to 303' 16" to 430'	430' - 480'	430'	Chlorination Aeration Sand Filtration
	1952	446'	24" to 80' 20" to 247'	247' - 446'	247'	Chlorination
	1955	507'	24" to 255' 16" to 314'	314' - 507'	?	Chlorination Aeration Sand Filtration
	1955	473'	16" to 289'	289' - 473'	?	Chlorination
	1955	500'	24" to 106' 16" to 315'	315' - 500'	315'	Chlorination Aeration Sand Filtration
	1961	1095'	24" to 103' 16" to 880'	880' - 1095'	?	Chlorination Aeration Sand Filtration

NON-RESPONSIVE

<u>Number & Address</u>	<u>Date</u>	<u>Well Depth</u>	<u>Casing</u>	<u>Openhole</u>	<u>Grouted</u>	<u>Treatment</u>
	1963	1095'	30" to 99' 24" to 270' 16" to 900'	900' - 1095'	900'	Chlorination Aeration Sand Filtration
	1964	1045'	30" to 95' 24" to 212' 16" to 891'	891' - 1045'	?	Chlorination Aeration Sand Filtration
	1965	485'	30" to 94' 24" to 253' 16" to 389'	389' - 485'	yes	Chlorination Aeration Sand Filtration
	1969	503'	30" to 102' 24" to 398'	398' - 503'	?	Chlorination Aeration Sand Filtration
	1973	500'	30" to 310' 24" to 425'	425' - 500'	yes	Chlorination Aeration Sand Filtration

NOTE: All wells are Fluoridated
 -Chlorination is with chlorine gas
 -Aeration is with air compressor
 -Filtration is with pressure sand

STORAGE CAPACITY

100,000 gallon elevated
 500,000 gallon elevated
 2,000,000 gallon underground
 1,500,000 gallon underground
 2 at 1,000,000 gallon elevated
 2 at 1,500,000 gallon ground

9,100,000 Gallons

Epidemiology

Epidemiologic investigations could provide valuable data relevant to human health assessment after exposure to PAH compounds in drinking water supplies. Questions such as dose after exposure, metabolic effects, and human health effects after chronic exposure need to be addressed. Environmental epidemiology requires an ordered and scientifically sound approach to achieve these results. Initial activity needs to be directed towards adequate exposure assessment.

Exposure Assessment

Exposure assessment involves the quantification, qualification, and temporal determination of exposure to the agent(s) of interest to the population and specific segments of the population.

- 1) Review water well pumping records and water supply distribution system history.

Records are maintained by MDH for gallons of water pumped per day for each well in SLP in conjunction with the addition of flouride. These records have been abstracted to provide monthly summaries of water pumpage of SLP drinking water wells (Table 4). This provides data on well utilization historically which aids in quantification of exposure and determination of probable service area of each well.

- 2) PAH analysis of SLP distribution system drinking water would provide data on current population exposure and shed light on drinking water treatment effects employed by SLP.

Wellhead PAH sampling has been preformed and continues to constitute the bulk of sampling conducted by MDH. Review of literature indicates that various treatments employed on SLP well water may reduce PAH levels in the distribution system. These treatments include chlorination, aeration, and sand filtration. However, it is not clear if the removal of PAH is the same for SLP drinking water (low turbidity) as has been reported to surface drinking waters and waste waters. Thus rigorous sampling needs to be conducted to determine these, and possibly other distribution system effects on PAH levels.

Experimental

SLP drinking water supply system is interconnected with treatments employed at the well head for each well. Treated water is then pumped into storage or distribution (Table Two issues need to be addressed in the sampling scheme: 1) What are the treatment effects on PAH levels found in SLP wellhead utilizing SLP drinking water treatment? and 2) What levels of PAH are the the residents of SLP presently exposed? This procedure will require the full cooperation of MDH and the full and active cooperation of SLP water supply officials. SLP water supply officials would need prior notification and two days of sampling (minimum) would be required.

Protocol SLP/PAH Distribution System Sample

- 1) Wells pumped at least million gallons per day during last 24 hours;
 - 2) Sample from cold water tap (domestic);
 - 3) Allow water to run for one minute prior to sampling;
 - 4) Ice or reffridgerate sample from time of collection until extraction. Extract ASAP.
- ***No contact with rubber seals, grease, or oil lubricated surfaces.

Sample Locations

Well # 15	Wellhead Postchlorination & Quench chlorine Post sand filtration & aeration & Quench Chlorine Quench chlorine @ +2 hours Quench chlorine @ +6 hours Quench chlorine @ +16 hours
Well # 7 & 9	Wellhead 7 Wellhead 9 Post Chlorination Distribution Distribution Distribution
Well # 5	Wellhead Post Chlorination Post Aeration Quench @ +2 hours Quench @ +6 hours Quench @ +16 hours
Well # 16	Wellhead Post chlorination Post sand filtration & aeration Distribution Distribution Distribution

**Note: Distribution samples need to be selected to minimize interference with other water supply wells and/or storage tanks.

- 3). Water well service area assessment needs to be performed due to the nature of the SLP municipal drinking water supply system and well contamination history. It may be assumed that different levels of PAH would be found in different areas of SLP based on the service area of highly contaminated water supply wells. It may be possible to utilize historical well usage data to determine the service area stilizing a surrogate marker parameter to indicate average well surface area during normal operating conditions.

Experimental

An acceptable surrogate would need the following characteristic:

- 1) Present at low, stable background levels;
- 2) Present at same levels for all wells;
- 3) Easily measured with high validity; and
- 4) Not harmful to the public.

Flouride is a candidate as a surrogate marker for PAH distribution. As flouride is added to all wells to meet state criteria, it would be necessary to have MDH approval for the temporary suspension of flouride addition in SLP. This temporary suspension (around one month) could be expected not to adversely effect the purpose of flouride addition.

- 1) Cease addition of flouride to all wells in SLP. Monitor water supply until levels reach background and are stable.
- 2) Utilizing historical pumpage data, pump wells are these levels. Add measured concentration of flouride to a single well and monitor service area. Alternate with all wells.

Discussion

Environmental epidemiology is especially hampered by insufficient or inappropriate exposure assessment with which to preform human health investigations. This tends to increase this misclassification which can diminish the observed relative risk if it is random. Although the SLP municipal water supply is contaminated with PAH, it does not necessarily follow that all residents of SLP are exposed at the same level. This exposure variability within SLP may be able to be characterized. Thus it is important to assess exposure areas and distribution system PAH concentrations.

Metabolic Effects

After exposure has been determined it becomes important to assess the possibility of a metabolic effect due to an exposure level. This is especially important with low exposures. A metabolic effect, if found, can support the contention that a health effect may occur under the exposure conditions. PAH compounds lend themselves to this as they are possibly stored in body fat and some PAH will induce enzyme activity. Measurement of bioaccumualtion is a means to quantify exposure. Measurement of PAH induced enzyme activity may be a means of determining whether sufficient exposure has occurred to lead to a health effect.

Mammalian tissues metabolize PAH to a large number of different compounds to make these substances sufficiently water soluble to be excreted. Aryl hydrocarbon monooxygenase is an almost ubiquitous enzyme system which is induced by some PAH. Nebert and Gelboin () found activity of this enzyme in 90% of the tissues examined. Aryl hydrocarbon monooxygenase has also been demonstrated in human tissues, including liver, lung, kidney, placenta, lymphocytes, monocytes, and alveolar macrophages. References concerning these issues are provided in appendix 1.

Review of Human Health Risk Assessment Documents Relevant to
St. Louis Park PAH Exposure in Drinking Water

Three human health risk assessment studies have been conducted relevant to PAH exposure in SLP municipal water supply. Two are literature based and one is an epidemiologic investigations which compare Third National Cancer Survey (TNCS) data for SLP with Minneapolis-St. Paul (Mpls-St. Paul) Standard Metropolitan Statistical Area (SMSA).

Human Health Risk Assessment

Human health risk due to PAH compounds present in drinking water has not been adequately investigated. The study by Dusich et al. (26) is limited by the use of the total SLP population as equally at risk, investigating a health risk prior to the presence of the suspected disease inducing agent (although evidence exist for longer exposure), and not using adequate control populations (SES, ethnicity differences in Richfield and Edina may be significant).

Future studies need to utilize more finely developed exposure criteria, justify the temporal relationship between disease and disease inducing agent. Control populations need to have adequate comparability and have evidence for no contamination of drinking water supply. Due to the unknown induction period for carcinogenesis due to low level PAH exposure, surveillance of the SLP population needs to continue so as to pick up these effects.

Health Risk Assessment Unit. 1977. Assessment of possible human health effects resulting from the contamination of the former Republic Creosote site. Minnesota Department of Health:Minneapolis. (35).

The report attempted to quantitatively assess existing and potential human health risk using currently available information. The evaluation is solely concerned with PAH induced carcinogenesis. A review of the properties of environmental PAH plus ambient exposure is presented. Animal bioassay and human epidemiologic data were reviewed. Both sets of data strongly implicate PAH as a carcinogen. Factors affecting carcinogenesis included repeated exposure of PAH at low levels. Tumor promotion and cocarcinogenic effects of phenol, anthralin, and creosote were reviewed Latent period was suggested to be dependent upon a dose time relationship.

The report concludes that there exists a significant potential impact on human health due to PAH levels found in SLP municipal water supply. Quantification or qualification of this impact could not be assessed due to limitations of available data.

Health Risk Assessment Unit. 1978. Health implication of polynuclear aromatic hydrocarbons in St. Louis Park drinking water. Minnesota Department of Health:Minneapolis. (36)

This report assess the risk associated with contamination of SLP drinking water supply with PAH. Municipal drinking water supplies may have significantly increased the intake of PAH compounds and this may pose a human health risk. This report also indicates that additional investigations be performed to better quantify and quality the extent of contamination and any human health risk.

The report concludes that wells 7,9,10 and 15 should be taken out of service. The use of SLP municipal drinking water supplies may have significantly increased the intake of pAH compounds and this may pose a human health risk. This report also indicates that additional investigations be performed to better quantify and quality the extent of contamination and any human health risk.

Dusich, K. et al. 1980. Cancer rates in a community exposed to low levels of creosote components in municipal water. Minnesota Med (Nov):803.

Incidence for 45 cancer sites or types were calculated for St. Louis Park, Edina, Richfield, and the Minneapolis-St. Paul SMSA using data from the Third National Cancer Survey, 1969-1971. Incidence was age-adjusted to SMSA male and female populations. Calculations included average annual age- and sex-specific cancer incidence, age-adjusted cancer incidence, standard incidence ratios, Mantel-Haenszel overall summary Chi-squares, and Z statistics. Population denominator was acquired from 1970 U.S. Census.

Male cancer incidence in SLP was not statistically significantly different from control areas. Female age-adjusted incidence for all sites combined (p .0005), breast cancer (p .0005), and cancers of the gastrointestinal tract (p .05) were higher in SLP than Edina, Richfield, and Mpls-St. Paul SMSA.

These elevated rates could not be attributed to PAH drinking water contamination. This is partly due to the design of the study which did not allow for information to be collected relevant to individuals of the sampled population. The individual characteristics of a population may explain an increased or decreased incidence of cancer when compared to control areas. Another limitation is the lack of adequate exposure assessment due to limitations of available data.

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Appendix 1: References

Polynuclear Aromatic Hydrocarbon Metabolism and Reproductive Effects

References: Polynuclear Aromatic Hydrocarbon Metabolism and Reproduction

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Appendix 2: Census Data

ST. LOUIS PARK, HENNEPIN COUNTY, MINNESOTA
MINNEAPOLIS - ST. PAUL SMSA

General Characteristics of the Population: 1970	Total SMSA	Total Henn Co	Edina	Richfield	St. Louis Park
<u>Age by Sex: Male</u>					
35 - 44	98 067	50 797	2 991	2 431	2 504
45 - 54	89 146	49 471	3 255	3 005	2 966
55 - 64	63 571	35 636	1 957	1 695	2 134
65 & Over	61 713	35 769	1 490	1 028	1 688
Total 35 & Over					
Total All Age	874 248	457 738	21 482	22 831	23 264
<u>Female</u>					
35 - 44	98 859	52 758	3 276	2 712	2 753
45 - 54	94 032	52 836	3 317	3 259	3 281
55 - 64	71 435	42 362	2 170	1 784	2 499
65 & Over	96 630	57 184	2 028	1 563	2 508
Total 35 & Over					
Total All Age	939 399	502 342	22 564	24 400	25 619
<u>Nativity, Parentage, & Country of Origin</u>					
Native of Native Parentage	1 492 020	774 186	35 675	39 235	36 486
Native of Foreign or Mixed Parentage	266 647	152 126	7 047	6 884	9 744
Foreign Born	54 980	33 768	1 292	1 118	2 663
<u>Years of School Completed</u>					
Median School Years Completed	12.4	12.5	14.3	12.6	12.6
Percent High School Graduates	66.1	67.9	88.7	78.1	75.8

	Total SMSA	Total Henn Co.	Edina	Richfield	St. Louis Park
<u>Residence in 1965</u>					
Same House as in 1970	862 805	448 087	20 883	25 445	24 920
<u>Occupation</u>					
Total Employed: 16 & Over	759 606	419 914	17 114	23 345	23 165
Female Employed: 16 & Over	306 480	175 565	5 386	10 000	9 551
Median Income	\$11 682	\$11 805	\$19 494	\$12 504	\$12 483
Mean Income	\$13 147	\$13 501	\$23 417	\$13 132	\$14 203
<u>Housing</u>					
With Public Water Supply	500 123	283 767	11 839	13 811	15 807
Housing Units	574 826	319 977	13 299	14 986	16 033
<u>Year Moved Into Unit</u>					
1968 - 1970	191 125	109 865	3 557	4 987	4 711
1965 - 1967	102 459	56 839	3 094	2 388	3 280
1960 - 1964	89 206	47 676	2 785	2 123	2 781
1950 - 1959	106 369	58 321	2 728	4 296	3 464
1949 or earlier	67 988	37 007	846	1 011	1 546

ST. LOUIS PARK, HENNEPIN COUNTY, MINNESOTA
MINNEAPOLIS - ST. PAUL SMSA

ST. LOUIS PARK - 1970 CENSUS TRACT DATA

	220.0	221.1	221.2	222.0	223.1	223.2	224.0	225.0	226.0	227.0	228.1	228.2	229.1	229.2	230.0
GENERAL POPULATION CHARACTERISTICS															
<u>Age by Sex: Male</u>															
35 - 44	44	149	183	388	130	197	257	195	135	188	104	102	157	119	156
45 - 54	42	178	197	373	128	215	330	230	190	254	206	162	167	103	191
55 - 64	27	108	113	137	64	139	173	123	149	215	163	206	184	112	221
65 & Over	18	54	73	46	37	230	124	64	106	191	99	238	169	77	162
Total 35 & Over															
Total All Age	342	1465	1382	2492	786	1884	2618	1674	1475	2140	1112	1355	1600	1016	1923
<u>Female</u>															
35 - 44	38	153	204	411	154	214	282	202	147	219	126	132	161	130	180
45 - 54	43	192	196	328	105	243	344	229	233	291	238	234	218	126	261
55 - 64	27	97	116	117	60	224	183	121	164	238	153	305	306	115	273
65 & Over	22	59	95	60	35	427	165	87	127	251	102	408	291	128	251
Total 35 & Over															
Total All Age	379	1482	1462	2577	808	2286	2750	1722	1548	2255	1166	1764	2070	1082	2268
<u>Nativity, Parentage, & Country of Origin</u>															
Native of Native Parentage	554	2261	1905	3842	1131	3220	4129	2936	2531	3276	1193	1805	2631	1633	3429
Native of Foreign or Mixed Parentage	102	548	756	1066	315	651	898	425	380	938	757	1054	845	262	747
Foreign Born	23	199	183	161	33	404	341	35	114	181	129	448	194	56	162
<u>Years of School Completed</u>															
Median School Years Completed	12.4	12.5	12.7	13.1	12.9	12.3	12.4	12.4	12.5	12.6	13.9	12.7	13.0	12.8	12.6
Percent High School Graduates	69.6	72.1	76.5	87.1	79.1	64.5	70.0	70.3	71.8	76.2	90.4	73.7	84.3	81.9	73.5

ST. LOUIS PARK - 1970 CENSUS TRACT DATA

GENERAL HOUSING CHARACTERISTICS

Residence in 1965

Same House as in 1970

OccupationTotal Employed: 16 & Over
Female Employed: 16 & OverMedian Income
Mean IncomeHousingWith Public Water Supply
Housing Units (Occupied)Year Moved Into Unit1968 - 1970
1965 - 1967
1960 - 1964
1950 - 1959
1949 or earlier

220.0	221.1	221.2	222.0	223.1	223.2	224.0	225.0	226.0	227.0	228.1	228.2	229.1	229.2	230.0
324	1604	1431	2869	986	1525	2805	2273	1718	2177	1322	1155	1663	1025	2043
310	1382	1394	2031	676	1987	2534	1482	1483	2193	969	1665	1842	908	2309
116	549	562	686	251	919	1046	562	619	908	300	766	843	383	1041
\$11647	\$11191	\$13563	\$14318	\$17707	\$11203	\$11429	\$12455	\$11959	\$12475	\$16739	\$13291	\$12681	\$13961	\$1128
\$12642	\$11766	\$14690	\$17638	\$19499	\$11419	\$11887	\$13051	\$12084	\$13313	\$23516	\$16287	\$13992	\$15277	\$120
218	897	959	1196	411	1425	1590	931	952	1531	660	1388	1414	632	1603
218	900	899	1274	415	1409	1597	952	952	1496	679	1370	1397	626	1598
88	263	292	254	44	565	522	140	197	517	79	537	462	121	630
24	203	232	342	76	413	337	172	152	275	117	330	292	115	200
64	177	143	339	177	168	269	151	75	273	124	249	176	120	276
23	214	221	324	106	244	350	384	326	275	241	104	260	118	274
19	43	11	15	12	19	119	105	202	156	118	150	207	152	218

Appendix 3

Third National Cancer Survey Data

Provided by: Dr. Deane Merrill
Lawrence Berkeley Laboratory

Accessed From:

Socio- Economic- Environmental- Data Integrated System (SEEDIS)
LBL Computer Science and Applied Mathematics Department
Part of Populations At Risk to Environmental Pollution (PAREP)
Funded by: Departments of Energy & Labor

ST. LOUIS PARK, HENNEPIN COUNTY, MINNESOTA
MINNEAPOLIS - ST. PAUL SMSA

THIRD NATIONAL CANCER SURVEY
1969-1971

-----TNCS.WH.Male Age Specific (10 Year)/ 3 Year Incidence per 100,000-----
TRACT # 220.00 221.01 221.02 222.00 223.01 223.02 224.00 225.00 226.00 227.00 228.01 228.02 229.01 229.02 230.00

35 - 44 Years

Stomach: 510-519
Colon excluding rectum: 531-539 544 (1)507
Rectum & rectosigmoid junction: 540-541
Lung, bronchus, & trachea: 620-624 (1)389 (1)512
Breast: 740-749
Bladder: 889

Total (1)507 (1)389 (1)512

45 - 54 Years

Stomach: 510-519 (2)1047
Colon excluding rectum: 531-539 544 (1)393 (1)599
Rectum & rectosigmoid junction: 540-541
Lung, bronchus, & trachea: 620-624 (1)507 (1)465 (1)303 (1)434
Breast: 740-749 (1)303
Bladder: 889 (1)485 (1)970 (1)523

Total (1)507 (1)465 (2)606 (1)434 (1)393 (1)485 (1)599 (1)970 (3)1570

55 - 64 Years

Stomach: 510-519 (1)813
Colon excluding rectum: 531-539 544 (1)730 (1)719 (1)465 (2)905
Rectum & rectosigmoid junction: 540-541 (1)1562 (1)719 (1)813 (1)465 (2)905
Lung, bronchus, & trachea: 620-624 (2)1851 (2)1770 (1)1562 (1)719 (3)1734 (1)813 (1)465 (1)485 (1)543 (2)905
Breast: 740-749
Bladder: 889 (2)1439 (1)465

Total (2)1851 (2)1770 (1)730 (2)3125 (5)3596 (3)1734 (3)2439 (4)1860 (1)485 (1)543 (6)2715

TNC.S.W.H. Male Age Specific (10 Year)/ 3 Year Incidence per 100,000															
TRACT #	220.00	221.01	221.02	222.00	223.01	223.02	224.00	225.00	226.00	227.00	228.01	228.02	229.01	229.02	230.00
65 Years & Over															
Stomach: 510-519			(1)1369				(1)806				(1)420	(1)592			(1)617
Colon excluding rectum: 531-539 544		(1)1852	(1)1369				(3)2419			(2)1052	(3)3030			(1)1299	(1)617
Rectum & rectosigmoid junction: 540-541															
Lung, bronchus, & trachea: 620-624	(1)5555	(1)1852	(1)1369				(2)870	(1)806	(1)1563	(1)943	(1)526	(1)1010		(1)592	(3)1852
Breast: 740-749															
Bladder: 889	(1)5555						(1)435	(1)806		(1)1886	(1)526		(4)1680		
Total	(2)11111	(2)3704	(3)4110				(3)1305	(6)4837	(1)1563	(2)2829	(4)2104	(4)4040	(5)2101	(2)1184	(1)1299(5)3085
35 Years & Over															
Incidence Rate	2	4	6	1	2	10	12	6	2	10	5	6	4	2	17
	1526	817	1060	106	557	1280	1354	980	345	1179	874	847	591	487	2328

ST. LOUIS PARK, HENNEPIN COUNTY, MINNESOTA
MINNEAPOLIS - ST. PAUL SMSA

THIRD NATIONAL CANCER SURVEY
1969-1971

	TNCs.WH.Female Age Specific (10 Year)/ 3 Year Incidence per 100,000													
TRACT #	220.00	221.01	221.02	222.00	223.01	223.02	224.00	225.00	226.00	227.00	228.01	228.02	229.01	229.02 230.00
<u>35 - 44 Years</u>														
Stomach 510-519														
Colon excluding rectum: 531-539 544														
Rectum & rectosigmoid junction: 540-541											(1)794			
Lung, bronchus, & trachea 620-624														
Breast: 740-749	(1)653	(2)980	(1)243	(2)1299			(2)709	(2)990						
Bladder: 889														
Total	(1)653	(2)980	(1)243	(2)1299			(2)709	(2)990			(1)794			
<u>45 - 54 Years</u>														
Stomach: 510-519			(1)510									(1)427		
Colon excluding rectum: 531-539 544														
Rectum & rectosigmoid junction: 540-541														
Lung, bronchus, & trachea 620-624				(1)304		(1)411	(1)290				(1)420			
Breast: 740-749	(1)510	(2)609				(1)411	(1)290	(1)436		(6)2062	(2)840	(3)1282	(1)458	(3)2380(2)766
Bladder: 889														
Total			(2)1020	(3)914		(2)822	(2)580	(1)436		(6)2062	(3)1260	(4)1609	(1)458	(3)2380(2)766
<u>55 - 64 Years</u>														
Stomach: 510-519			(1)854					(1)609				(1)326		
Colon excluding rectum: 531-539 544			(1)854			(2)1093	(1)826		(1)653		(1)326		(1)366	
Rectum & rectosigmoid junction: 540-541													(1)366	
Lung, bronchus, & trachea: 620-624	(1)862		(1)1667	(1)446		(1)826								
Breast: 740-749	(4)3448	(2)1709		(3)1339	(1)546				(3)1251	(1)653	(5)1639	(3)980	(3)2609	(8)2930
Bladder: 889				(1)446					(1)420	(1)653				
Total	(5)4310	(4)3418	(1)1667	(5)2232	(3)1639	(2)1652	(1)609	(4)1671	(3)1959	(5)1639	(5)1634	(3)2609	(10)3663	

TNCs.W.H.Female Age Specific (10 Year)/ 3 Year Incidence per 100,000															
TRACT #	220.00	221.01	221.02	222.00	223.01	223.02	224.00	225.00	226.00	227.00	228.01	228.02	229.01	229.02	230.00
65 Years & Over															
Stomach: 510-519						(1)234					(2)490			(1)398	
Colon excluding rectum: 531-539 544			(1)1053			(5)1171	(4)2424	(1)1194	(2)796	(1)980	(7)1715	(4)1375	(1)781	(2)797	
Rectum & rectosigmoid junction: 540-541		(1)1694				(3)703		(1)1194	(1)787		(2)490	(1)343			
Lung, bronchus, & trachea: 620-624						(1)234			(3)1195	(1)980	(2)490			(1)398	
Breast: 740-749			(1)1053			(2)468	(2)1212	(3)3478	(3)2362	(2)796	(2)1960	(6)1470	(2)687	(1)781	(4)1593
Bladder: 889			(1)1053				(1)606				(1)245			(2)797	
Total		(1)1694	(3)3159				(12)2810	(7)4242	(5)5747	(4)3049	(7)2788	(4)3921	(22)4901	(7)2405	(2)1562(10)3783
35 Years & Over Total	2	12	8	3	19	14	10	6	17	11	29	13	8	23	
Incidence Rate	399	1964	873	848	1714	1437	1564	894	1701	1777	2688	1332	1603	2383	